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SARS-CoV-2 infection in a psoriatic patient treated with IL-23 inhibitor

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Since December 2019, an outbreak of 2019 novel coronavirus disease (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has been spreading worldwide. This has risen concern among patients undergoing biologics and physicians who administer them, as far as the possible increase of incidence and severity of COVID-19 in this delicate population concerns¹.

We describe the case of a 32-year-old woman, affected by psoriasis and psoriatic arthritis since 18 years, previously treated with several conventional and biologic drugs, including cyclosporine, methotrexate, infliximab, etanercept, adalimumab, secukinumab and ixekizumab. She had no other medical conditions.

In April 2019 she developed a severe Crohn's disease while taking ixekizumab. Therefore, she switched to ustekinumab, with improvement of Crohn's disease but a worsening of both psoriasis and psoriatic arthritis. On 6th November, we added methotrexate 10 mg/week, which was further increased to 25 mg/week after 4 weeks because of an unsatisfactory response. On 23rd December, since psoriasis was still worsening, we switched ustekinumab to guselkumab, while maintaining methotrexate at 25 mg/week.

On February 26th, after two injections of guselkumab the patient showed a marked improvement of psoriasis and arthritis.

On February 29th, she went out for dinner with some friends and, 2 days later, one of them was discovered to be affected by COVID-19. On March 4th she had mild rhinorrhea and fever (37.4 °C) and the next day she was tested positive for SARS-CoV-2.

The day after the body temperature lowered to 36.3 °C and the rhinorrhea was still mild. We advised her to interrupt methotrexate and to postpone the next guselkumab injection, which was originally scheduled for March 16th.

In the following days, the body temperature never rose above 36,5 °C and she never developed sore throat, cough, shortness of breath or other symptoms of the infection. Her blood tests revealed increased erythrocyte sedimentation rate (120 mm/h), C-reactive protein (4,76 mg/dL), D-dimer (381 ug/L) and fibrinogen (701 mg/dL). All the other parameters were normal.

On March 13th the rhinorrhea subsided. On March 20th RT-PCR was still positive for SARS-CoV-2. On March 28th and March 30th the tests resulted negative, meeting the criteria to be considered successfully healed.

In COVID-19, inflammatory cytokines assume a double role: firstly, they stimulate the activation of an effective immune response, while later they can mediate the development of an exaggerated systemic inflammation. This “cytokine storm” is both ineffective toward the pathogen and detrimental for the body, eventually leading to acute respiratory distress syndrome and potentially to death².

Available data suggest that the adaptive response toward SARS-COV-2 develops mainly in a Th1 polarized fashion, being CD8⁺ cytotoxic cells the main effectors of the antiviral response². With the progression of the disease, the worsening of clinical conditions is associated to a marked increase in proinflammatory cytokines, such as IL-1, IL-6 and TNF alfa^{2,3}.

Interestingly, the IL-23/IL-17 axis does not seem to be pivotal in an effective immune response. On the contrary, observations carried on both coronavirus and non-coronavirus pneumonia patients show that an aberrant Th17 polarization may correlate with a worse outcome^{4,5}.

Based on these observations, a clinical trial investigating the use of ixekizumab associated to antiviral therapy is currently ongoing in China as a possible treatment for COVID-19 infection⁶.

In conclusion, we reported the first case of COVID-19 infection in a psoriatic patient treated with a biologic. The outcome of this case and data from currently available literature suggest that IL-23/IL-17 axis inhibition might not be detrimental in the setting of COVID-19 infection. Further data are needed to support this hypothesis.

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